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1995-1996 INFLUENZA SEASON: CANADIAN LABORATORY DIAGNOSES AND STRAIN CHARACTERIZATION

Introduction

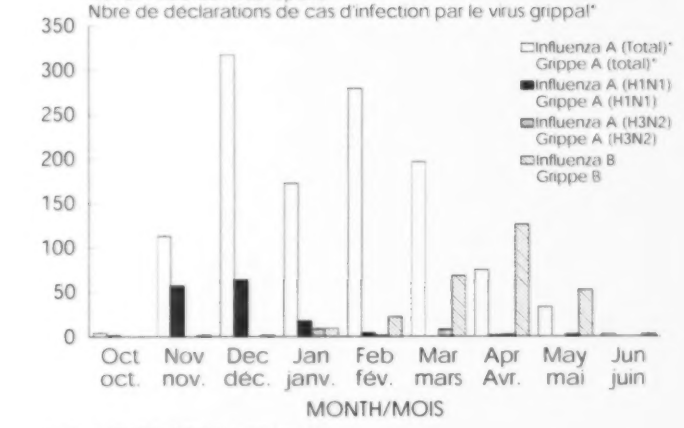
In collaboration with the World Health Organization (WHO), international collaborating laboratories, provincial laboratories, and other Canadian hospital and university-based virus laboratories, the Laboratory Centre for Disease Control (LCDC) conducts national surveillance on human influenza viruses. This surveillance monitors influenza activity, detects and describes antigenic changes in circulating strains of influenza virus in Canada and, through periodic serosurveys, estimates susceptibility to currently circulating and emerging strains. Canadian influenza surveillance information and actual representative strains are then shared with the WHO's collaborating centres for influenza to contribute to global influenza monitoring.

Influenza Activity

Figure 1 shows the number and month of laboratory-confirmed influenza virus isolations, detections, and serodiagnoses reported from 32 of the 39 laboratories that contribute to the Canadian Virus Reporting (CVR) program, a surveillance program covering all laboratory-diagnosed viral diseases. The influenza season in Canada began in late November 1995 and continued to late June 1996. In addition, at least one influenza isolate was reported each month from July to October 1995.

There were 281 reports of influenza B from November 1995 through June 1996 with the largest number (126) occurring in April. One thousand, one hundred and ninety laboratory reports of

Figure 1
Laboratory evidence of human influenza virus infections in Canada, 1995-1996 season



* includes A(H1N1), A(H3N2), and non-typed
inclut A(H1N1), A(H3N2) et des souches non typées

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influenza A and influenza A subtypes were received in the same period. The data from the Respiratory Virus Surveillance Program established by the Bureau of Infectious Diseases, LCDC, are in good agreement with what are shown in Figure 1.

Strain Characterization

Table 1 indicates the provincial source and identity of submitted isolates that were strain-typed at LCDC, while Figure 2 shows the change in influenza virus strains by month of submission to LCDC as the season progressed. Most influenza A strains received early in the season were A/Texas/36/91 (H1N1)-like viruses whereas A/Johannesburg/33/94(H3N2)-like strains accounted for most of the late-season influenza A isolates. A/Taiwan/1/86(H1N1)-like strains also were identified early in the season. Isolates resembling antigenic variant A/Wuhan/359/95 (H3N2) appeared later in April and May 1996. Two of the A/Wuhan/359/95-like isolates were later characterized at the Centers for Diseases Control and Prevention as similar to A/Alaska/ 10/95, a strain related to A/Wuhan/359/95. All the influenza B isolates received were B/Beijing/184/93-like strains, the majority of which arrived in April 1996.

Figure 3 shows the distribution of influenza A virus strains received at LCDC by month from different regions of Canada as the season progressed. Isolates of all four strains were received first from the Prairie provinces. In addition, A/Wuhan/359/95(H3N2)-like strains appeared in all regions except the Atlantic provinces in April 1996, suggesting that either the virus entered different regions in Canada simultaneously or it spread very rapidly once introduced into the country.

Discussion

In Canada, the 1995-1996 influenza season came earlier and was over sooner than the previous one[1]. The predominating virus strains were antigenically similar to vaccine component strains[2]. A/Taiwan/1/86-like strains had been the predominant H1N1 isolates in Canada from the 1987-1988 season to the

Figure 2
LCDC antigenic characterization completed on influenza virus isolates in the 1995–1996 season by month of submission

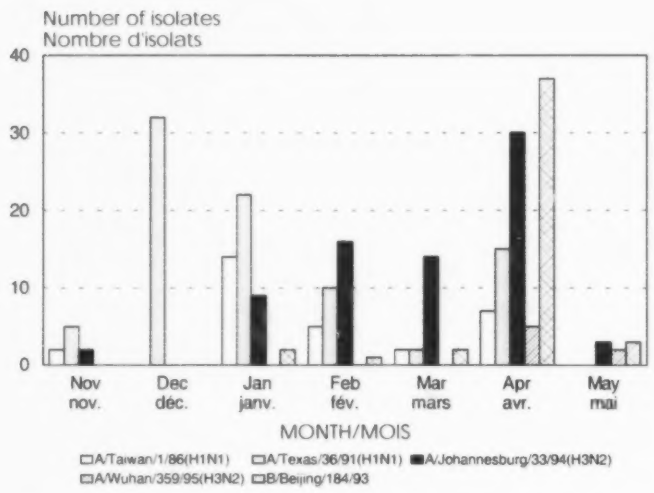


Table 1
LCDC strain characterization completed on influenza isolates in Canada (submitted from 22 November 1995 to 28 June 1996)

INFLUENZA	PROVINCE										
	Nfld	PEI	NS	NB	Que	Ont	Man	Sask	Alta	BC	Total
TYPE A (H1N1)											
A/TAIWAN/1/86-like			1	1	4	6	7	5	2	3	29
A/TEXAS/36/91-like*			3		25	20	11	13	3	12	87
TYPE A (H3N2)											
A/JOHANNESBURG/33/94-like**	3		6	4	24	13	3	4	10	5	72
A/WUHAN/359/95-like***					3	1	3			2	9
TOTAL A	**3**		**10**	**5**	**56**	**40**	**24**	**22**	**15**	**22**	**197**
TYPE B											
B/BEIJING/184/93****					6	14	5	6	6	5	42
TOTAL B					**6**	**14**	**5**	**6**	**6**	**5**	**42**
TOTAL A and B	3		**10**	**5**	**62**	**54**	**29**	**28**	**21**	**27**	**239**

* A/Texas/36/91 is the (H1N1) component of the 1995-1996 vaccine and A/Taiwan/1/86 is very closely related to A/Texas/36/91.
** A/Johannesburg/33/94-like virus was the influenza A(H3N2) vaccine component in the 1995-1996 influenza vaccine.
*** A/Wuhan/359/95-like virus is the WHO recommended influenza A(H3N2) component of the 1996-1997 influenza vaccine.
**** B/Beijing/184/93-like virus is the WHO recommended influenza B component of the 1995-1996 influenza vaccine and is antigenically indistinguishable from B/Harbin/7/94.

1991-1992 season and reappeared along with A/Texas/36/91-like strains in the 1995-1996 season (Table 1); however, antigenically A/Taiwan/1/86-like strains are closely related to the A/Texas/36/91-like vaccine component. B/Beijing/184/93 was the influenza B component of the 1995-1996 influenza vaccine, as recommended by WHO, and is antigenically indistinguishable from B/Harbin/7/94.

Influenza A(H3N2), A(H1N1), and B viruses continued to circulate worldwide[3]. Although most influenza A isolates were similar to A/Johannesburg/33/94, the influenza A(H3N2) vaccine component in the 1995-1996 influenza vaccine, an increasing number of recently isolated A(H3N2) strains from Asia, Europe, and North America are similar to the antigenic variant A/Wuhan/359/95. Virtually all influenza A(H1N1) viruses (98%) that have been antigenically characterized were similar to the reference strains A/Taiwan/1/86 and A/Texas/36/91[3]. Antigenically characterized influenza B viruses isolated recently in Asia, Europe, and the United States were similar to the reference strains B/Beijing/184/93 and B/Harbin/7/94 which are antigenically indistinguishable[3].

The 1995-1996 season experience in Canada was similar to the global picture. The National Advisory Committee on Immunization has recommended that the trivalent vaccines for the 1996-1997 season contain an A/Wuhan/359/95(H3N2)-like strain, an A/Texas/36/91(H1N1)-like strain, and a B/Beijing/184/93-like strain[4].

Acknowledgements

The collaboration of laboratories in the CVR program and of provincial and hospital laboratories that forwarded early and representative isolates of influenza virus is a vital part of influenza surveillance in Canada.

Influenza virus isolates were submitted from the following centres:

British Columbia Centre for Disease Control, Virology Services, Vancouver, BC;

Provincial Laboratory of Public Health for Southern Alberta, Calgary, Alberta;

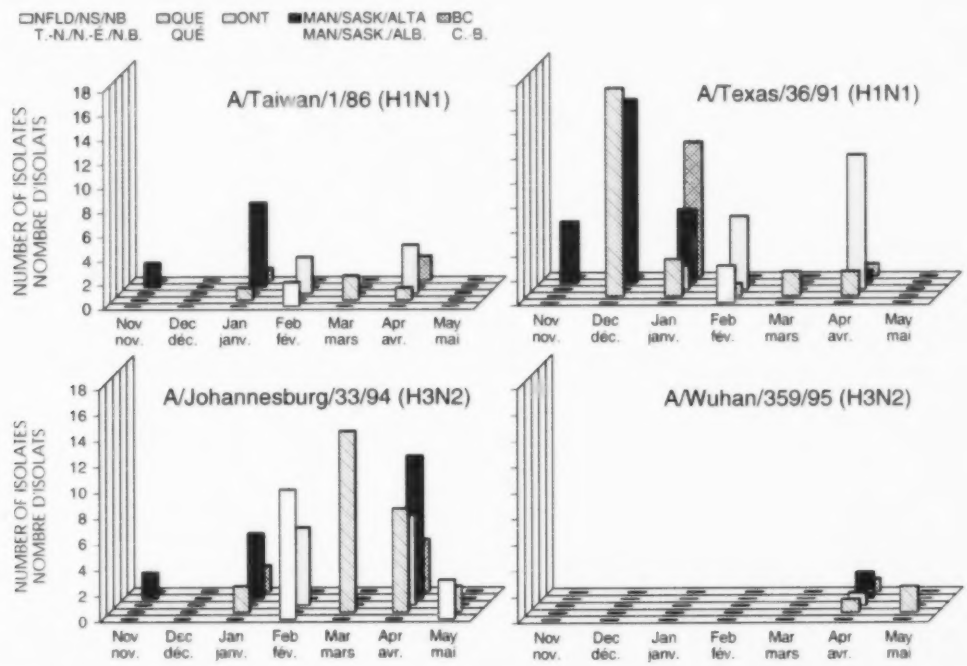
Saskatchewan Public Health Laboratory, Laboratory and Disease Control Services Branch, Regina, Saskatchewan;

Cadham Provincial Laboratory, Winnipeg, Manitoba;

Regional Public Health Laboratory, Laboratory Services Branch, Virus Laboratory, Toronto, Ontario;

Laboratoire de santé publique du Québec, Sainte-Anne-de-Bellevue, Québec;

Figure 3
Influenza A virus isolates characterized at LCDC by month of submission and region



Hôpital G.L. Dumont, Moncton, New Brunswick;

Victoria General Hospital, Halifax, Nova Scotia;

Provincial Public Health Laboratory, St. John's, Newfoundland.

Carol Stansfield of LCDC conducted the influenza strain typing.

References

1. Weber JM. *1994-1995 Influenza season: canadian laboratory diagnoses and strain characterization.* CCDR 1995;21:181-85.
2. National Advisory Committee on Immunization. *Statement on influenza vaccination for the 1995-96 season.* CCDR 1995;21:105-13.
3. CDC. *Update: Influenza activity - United States and Worldwide, 1995-96 season, and composition of the 1996-97 influenza vaccine.* MMWR 1996;45:326-29.
4. National Advisory Committee on Immunization. *Statement on influenza vaccination for the 1996-97 season.* CCDR 1996;22:89-97.

Source: *S Zou, PhD, J Weber, PhD, Surveillance, Influenza and Viral Exanthemata, National Laboratory for Special Pathogens, Bureau of Microbiology, LCDC, Ottawa, Ontario.*

International Notes

ANTIGENIC ANALYSIS OF RECENT INFLUENZA VIRUS ISOLATES AND INFLUENZA ACTIVITY IN THE SOUTHERN HEMISPHERE

Since the influenza vaccine recommendations for the 1996-1997 season were issued, influenza virus isolates from Africa, the Americas, Asia, Europe, and Oceania have been characterized antigenically at the WHO Collaborating Centres.

Influenza A virus isolates predominated. Both influenza A(H3N2) and A(H1N1) viruses were isolated in countries of Asia, Europe, and North America. Influenza B virus isolates, few in number during the peaks of influenza activity in December and January, increased during February and March. The majority of influenza viruses isolated in the northern hemisphere were antigenically closely related to the recommended vaccine strains for the 1995-1996 season. The majority of influenza A(H3N2) virus isolates were similar to A/Johannesburg/33/95; the proportion of isolates similar to A/Wuhan/359/95 increased during 1996. Most influenza A(H1N1) virus isolates were similar to A/Singapore/6/86 and A/Texas/36/91; viruses similar to an antigenically distinct variant represented by A/Wuhan/371/95 were isolated only in China and Hong Kong. The majority of influenza B virus isolates were similar to B/Beijing/184/93 and B/Harbin/7/94.

Influenza activity in the southern hemisphere has generally been moderate to severe. New Zealand, in particular, experienced a severe epidemic. Influenza A(H3N2) predominated in most countries causing outbreaks in Argentina, Australia, Chile, Madagascar, New Zealand, and South Africa. Virus isolates were also reported in Brazil and Senegal. The majority of A(H3N2) virus isolates were antigenically similar to A/Wuhan/359/95. Outbreaks due to influenza A(H1N1) occurred in Chile and South Africa. The incidence of influenza B was low.

Source: *WHO Epidemiological Record, Vol 71, No 39, 1996.*

SURVEILLANCE FOR CREUTZFELDT-JAKOB DISEASE — UNITED STATES

In the United States, deaths associated with Creutzfeldt-Jakob disease (CJD) among persons aged < 30 years have been extremely rare (median age at death: 68 years). As a result of the newly recognized variant of CJD described in the United Kingdom, the Centers for Disease Control and Prevention (CDC) updated their previous review of national CJD mortality and began conducting active CJD surveillance in five sites. These reviews did not detect evidence of the occurrence of the newly described variant form of CJD in the United States.

Based on multiple cause-of-death data obtained from CDC's National Center for Health Statistics, the annual death rates for CJD during 1979-1994 were stable at approximately one case per million population (Figure 1). Data for 1979-1993 are final; 1994 data are provisional.

The number of deaths attributed to CJD among persons aged < 45 years ranged from zero in 1984 to 8 in 1981 and 1993. In most years no CJD-associated deaths were reported among persons aged < 30 years: no year had more than one death. During 1990-1994, CJD was coded as a cause of death on the death certificate for two persons aged < 30 years. One of them died in 1993 and had been previously identified as part of ongoing surveillance for CJD among recipients of pituitary-derived human-growth hormone; the other died in 1994, but was excluded from analysis because follow-up investigation revealed a postmortem examination that did not confirm the initial CJD diagnosis but indicated a diffuse T-cell proliferative disease.

Active CJD surveillance

In early April 1996, active surveillance for the newly reported variant of CJD and physician-diagnosed CJD cases was conducted in four sites (Connecticut, Minnesota, Oregon, and the San Francisco Bay area of California), as well as the Division of Public Health, Georgia Department of Human Resources, including the Atlanta Metropolitan Active Surveillance Project (total 1993 population for theses areas: 16.3 million). CJD deaths were defined as any deaths that the surveillance teams in each of these five sites identified as having been attributed to CJD by a physician. Surveillance efforts included review of available death certificate data during 1991-1995 and contact by phone, mail, or fax with neurologists, neuropathologists, and pathologists to identify patients who died from CJD during 1991-1995. Approximately 800 neurologists and neuropathologists, constituting 92% to 100% of these specialists in the surveillance areas, and over 90% of pathologists in three areas were contacted. In addition, clinical and neuropathological records for each CJD patient aged < 55 years were sought for review.

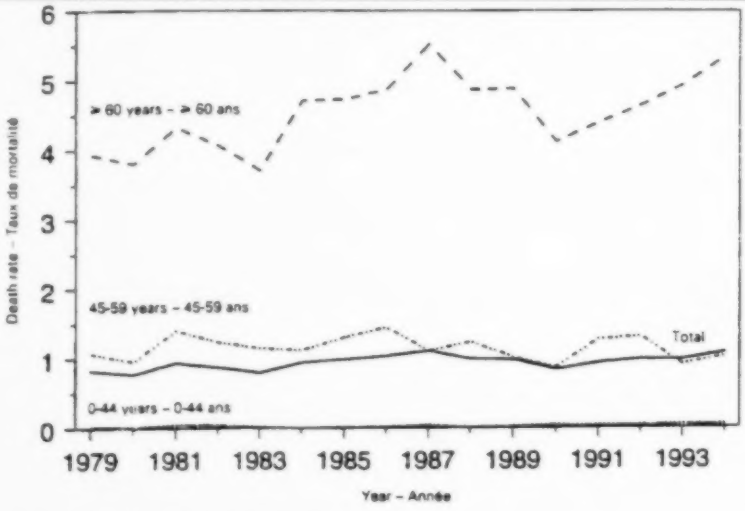
A total of 94 deaths attributed to CJD were identified in the active surveillance areas during 1991-1995. The annual number of CJD deaths was stable (mean: 19; range: 18 to 19), and the average annual CJD death rate was 1.2 (range by site: 0.7 to 1.7) per million population. (Table 1). Consistent with the national CJD mortality pattern nine (10%) of the 94 patients were aged < 55 years; one of the nine was aged < 45 years, and none were aged < 30 years.

The clinical and neuropathological record review of the nine patients aged < 55 years did not identify any with the variant form of CJD. A brain biopsy was performed for the one case who was aged < 45 years, and an autopsy was performed for four of the other eight. One case for whom there was no brain biopsy or autopsy was from a family that had a known genetic abnormality associated with CJD.

One additional CJD patient aged < 45 years who died in early 1996 was identified by the surveillance teams. This patient's clinical history was similar to the description of the new variant of CJD, but brain pathology at autopsy was inconsistent with that diagnosis.

Of the 94 CJD deaths, 81 (86%) were identified from death certificate review. For the 13 deaths that were identified only through survey of neurologists, neuropathologists, or pathologists, the death certificate either was not coded as CJD or had not yet been filed.

Figure 1
Age adjusted and age-specific death rates[a] for Creutzfeldt-Jakob disease, United States, 1979-1994[b]



[a] Per million population. - Par million d'habitants.
[b] Data for 1994 are provisional. - Les données pour 1994 sont provisoires.

Table 1
Number of deaths from Creutzfelft-Jakob disease, by year and age group and average annual death rate[a], by age group, active surveillance sites[b], United States, 1991-1995

Year	Age group (years)		
	< 55	≥ 55	Total
1991	2	17	19
1992	2[c]	17	19
1993	1	17	18
1994	1	18	19
1995	3	16	19
Total	**9**	**85**	**94**
Rate	0.1	5.3	1.2

[a] Per million population.
[b] Connecticut, Minnesota, Oregon, and the San Francisco Bay area of California, as well as the Division of Public Health, Georgia Department of Human Resources, including the Atlanta Metropolitan Active Surveillance Project (total 1993 population for these areas: 16.3 million).
[c] One case occurred in a person aged under < 45 years.

Source: *WHO Epidemiological Record, Vol 71, No 36, 1996.*

CREUTZFELDT-JAKOB DISEASE — SWITZERLAND

A total of 43 cases of Creutzfeldt-Jakob disease (CJD) were notified to the Swiss Federal Office of Public Health for the period 1991 to 1995. During this period, the incidence of CJD remained stable, i.e. an average of 1.2 cases per million population. In 80% of the cases, the diagnosis was confirmed by autopsy; 95% of the patients are known to have died. The mean age of patients was 66 years, half of them (49%) being males. The youngest person was 43 years old and died in 1992. The incidence of CJD in Switzerland is comparable with that of other European countries such as France, Germany, Italy, the Netherlands and the United Kingdom.

Discussion regarding possible transmission of bovine spongiform encephalopathy has intensified since 20 March 1996, when the British authorities officially declared 10 cases of a variant of CJD among persons aged < 42 years. No cases of disease corresponding to this new variant of CJD have yet been notified to the Swiss Federal Office of Public Health.

Source: *WHO Weekly Epidemiological Record, Vol 71, No 32, 1996.*

Announcement

LCDC LAUNCHES NEW WEBSITE: http://www.hwc.ca/hpb/lcdc

The LCDC Website, launched on October 24, 1996, is a key Canadian public health site. It contains disease surveillance and control information, disease prevention guidelines, health hazard advisories and travel health information. LCDC publications from 1995 onward, including Chronic Diseases in Canada, AIDs in Canada, the Canadian Hospitals Injury Reporting and Prevention Program (CHIRPP) News, Canada Communicable Disease Report, Measles Update, Farm Family Health, and descriptions of LCDC's epidemiology and laboratory programs.

The site also provides links to other public health sites in Canada and to international public health bulletins. It can be thought of as a digital library — a collection of documents available to anyone with Internet access. Visit our site for a variety of public health information.

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